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=> s (growth(w)hormone or GH or hGH) and (multiple(w)system(w)atrophy or MSA)
L1          262 (GROWTH(W) HORMONE OR GH OR HGH) AND (MULTIPLE(W) SYSTEM(W) ATRO
                PHY OR MSA)

=> s (growth(w)hormone or GH or hGH) and multiple(w)system(w)atrophy
L2           96 (GROWTH(W) HORMONE OR GH OR HGH) AND MULTIPLE(W) SYSTEM(W) ATROP
                HY

=> s l2 and (subcutaneous or intramusc?)
L3            4 L2 AND (SUBCUTANEOUS OR INTRAMUSC?)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4            3 DUP REM L3 (1 DUPLICATE REMOVED)

=> s l2 and py<2003
L5           46 L2 AND PY<2003

=> dup rem l5
PROCESSING COMPLETED FOR L5
L6           17 DUP REM L5 (29 DUPLICATES REMOVED)

=> dis ibib abs l4 1-3

L4   ANSWER 1 OF 3  EMBASE  COPYRIGHT (c) 2010 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:    2007344844  EMBASE
TITLE:               Safety and tolerability of growth hormone
                     therapy in multiple system
                     atrophy: A double-blind, placebo-controlled study.
AUTHOR:              Holmberg, Bjorn, Dr. (correspondence); Johansson, Jan-Ove
CORPORATE SOURCE:    Movement Disorders Unit, Sahlgrenska University Hospital,
                     Goteborg University, Sweden. bjorn.holmberg@neuro.gu.se
AUTHOR:              Poewe, Werner; Wenning, Gregor
CORPORATE SOURCE:    Department of Neurology, University Hospital Innsbruck,
                     Innsbruck, Austria.
AUTHOR:              Quinn, Niall P.; Mathias, Chris
CORPORATE SOURCE:    Sobell Department of Motor Neuroscience and Movement
                     Disorders, Institute of Neurology, London, United Kingdom.
AUTHOR:              Tolosa, Eduardo; Cardozo, Adriana
CORPORATE SOURCE:    Hospital Clinic de Barcelona, Servicio de Neurologia,
                     Barcelona, Spain.
```

AUTHOR: Dizdar, Nil
CORPORATE SOURCE: Department of Neurology, Linkoping University Hospital, Sweden.
AUTHOR: Rascol, Olivier; Slaoui, Tarik
CORPORATE SOURCE: Department of Pharmacology, Clinical Investigation Center, Hopital Purpan, Toulouse, France.
AUTHOR: Rascol, Olivier; Slaoui, Tarik
CORPORATE SOURCE: Department of Neurosciences, Clinical Investigation Center, Hopital Purpan, Toulouse, France.
AUTHOR: Holmberg, Bjorn, Dr. (correspondence)
CORPORATE SOURCE: Sahlgrenska University Hospital, Goteborg University, SE 41345 Goteborg, Sweden. bjorn.holmberg@neuro.gu.se
SOURCE: Movement Disorders, (15 Jun 2007) Vol. 22, No. 8, pp. 1138-1144.
Refs: 23
ISSN: 0885-3185; E-ISSN: 1531-8257 CODEN: MOVDEA
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 037 Drug Literature Index
038 Adverse Reactions Titles
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 3 Aug 2007
Last Updated on STN: 3 Aug 2007

AB The objective of this study was to investigate tolerability and possible neurotrophic effects of growth hormone (GH) in treatment of multiple system atrophy (MSA). In this double-blind pilot study, MSA patients were randomized to recombinant human growth hormone (r-hGH, n = 22), 1 mg every second day (6 months) followed by alternating daily injections of 1 mg and 0.5 mg (6 months), or matched placebo (n = 21). Safety analysis demonstrated no obvious between-group differences. In both groups, there was progressive worsening of Unified Parkinson's Disease Rating Scale total score, which tended to be less in r-hGH -treated patients (12.9% at 6 months, 25.3% at 12 months) than in placebo (17.0% and 35.7%). Similarly, there was a trend to less worsening in Unified MSA Rating Scale total score with r-hGH (13.2% and 21.2%) than with placebo (21.1% and 36.5%). Cardiovascular reflex autonomic testing also tended to show less deterioration with r-hGH than with placebo at 12 months. However, 95% CI did not indicate treatment differences for any efficacy measures. In conclusion, r-hGH administration in MSA patients for up to 1 year appears safe and might influence disease symptoms, signs and, possibly, progression. The results support further studies utilizing higher doses in more patients. .COPYRGT. 2007 Movement Disorder Society.

L4 ANSWER 2 OF 3 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2004551521 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15390061
TITLE: Levodopa treatment does not affect low-dose apomorphine test in patients with Parkinson's disease.
AUTHOR: Happe Svenja; Tings Tobias; Helmschmied Kathrin; Neubert Karin; Wuttke Wolfgang; Paulus Walter; Trenkwalder Claudia
CORPORATE SOURCE: Department of Clinical Neurophysiology, University of Gottingen, Germany.. shappe@gwdg.de
SOURCE: Movement disorders : official journal of the Movement Disorder Society, (2004 Dec) Vol. 19, No. 12, pp. 1511-5.
Journal code: 8610688. ISSN: 0885-3185. L-ISSN: 0885-3185.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)

(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 4 Nov 2004
Last Updated on STN: 8 Apr 2005
Entered Medline: 7 Apr 2005

AB Challenge with low-dose apomorphine causes a significant rise in growth hormone (GH) in patients with Parkinson's disease (PD) compared to controls and patients with multiple system atrophy (MSA) who have not previously received dopaminergic treatment. To date, it has not been demonstrated whether an apomorphine-induced rise in GH can still be detected in PD patients who are currently treated with levodopa. We investigated whether an ongoing treatment with levodopa influences the GH response to subcutaneously applied low-dose apomorphine in PD patients. We studied 44 patients with idiopathic PD using the low-dose apomorphine test. Twenty-three patients were under treatment with levodopa and 21 patients were without any dopaminergic therapy. GH and cortisol levels were analyzed at time of injection and 45 minutes and 60 minutes after subcutaneous apomorphine injection. Forty-five minutes after apomorphine injection, there was no significant difference between the mean rise in plasma GH in untreated PD patients compared with levodopa-treated patients (P = 0.235). There was no increase of cortisol levels in each treatment group. Age, sex, duration, and severity of the disease did not show a covariate effect with GH levels. A small group of PD patients (n = 8) treated with dopamine agonists and a small group of patients with MSA (n = 5) as well as patients with vascular parkinsonism (n = 5) did not show any increase of GH. Our data suggest that the apomorphine-induced rise in GH does not depend on previous levodopa treatment in PD patients but, as expected, is blocked by dopamine agonists and is not present in patients with other than idiopathic parkinsonian syndrome. Thus, the low-dose apomorphine test may also be a useful biological marker in the early differential diagnosis of PD patients who have already received levodopa treatment.
2004 Movement Disorder Society.

ACCESSION NUMBER: 2001072402 EMBASE
TITLE: Increased growth hormone response to apomorphine in Parkinson disease compared with multiple system atrophy.
AUTHOR: Friess, E., Dr. (correspondence); Kuempfel, T.; Winkelmann, J.; Schmid, D.; Uhr, M.; Rupprecht, R.; Holsboer, F.; Trenkwalder, C.
CORPORATE SOURCE: Max Planck Institute of Psychiatry, Kraepelinstr 10, D-80804 Munich, Germany. friess@mpipsykl.mpg.de
SOURCE: Archives of Neurology, (2001) Vol. 58, No. 2, pp. 241-246.
Refs: 17
ISSN: 0003-9942 CODEN: ARNEAS
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 003 Endocrinology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 8 Mar 2001
Last Updated on STN: 8 Mar 2001

AB Background: Parkinson disease (PD) is often difficult to distinguish from

parkinsonian syndromes of other causes in early stages of the disease. In search of a suitable endocrinologic challenge test, we investigated do paminergic sensitivity in patients with de novo parkinsonian syndromes. Objective: We measured the growth hormone (GH) response to a subthreshold dose of the dopamine 1-dopamine 2 receptor agonist apomorphine hydrochloride to differentiate parkinsonian syndromes from PD. Patients and Methods: Seventeen patients with a clinical diagnosis of PD, 16 patients with a clinical diagnosis of multiple system atrophy, and 11 healthy controls. The GH response to a subthreshold dosage of apomorphine and to somatorelin (GH-releasing factor) was tested in a randomized order; on the third day the protocol was repeated with a clinically effective dose of apomorphine. Results: The GH response to the low dose of apomorphine was significantly increased in patients with PD when compared with patients with multiple system atrophy or the control subjects (multivariate analyses of covariance; univariate F test, all P<.05). In contrast, there were no significant group differences with use of the higher dose of apomorphine or in the somatorelin-induced GH release. Conclusions: The GH response to a subthreshold dose of apomorphine appears to be a useful tool to identify patients with PD vs multiple system atrophy. The enhanced GH response to a subthreshold dopaminergic stimulus may reflect a hypersensitivity of the extrastriatal dopamine receptors in PD.

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L1           262 S (GROWTH(W)HORMONE OR GH OR HGH) AND (MULTIPLE(W)SYSTEM(W)ATRO
L2            96 S (GROWTH(W)HORMONE OR GH OR HGH) AND MULTIPLE(W)SYSTEM(W)ATROP
L3             4 S L2 AND (SUBCUTANEOUS OR INTRAMUSC?)
L4             3 DUP REM L3 (1 DUPLICATE REMOVED)
L5            46 S L2 AND PY<2003
L6            17 DUP REM L5 (29 DUPLICATES REMOVED)

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=> dis ibib abs l6 1-17
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L6   ANSWER 1 OF 17     MEDLINE on STN                  DUPLICATE 1
ACCESSION NUMBER:    2002703255      MEDLINE
DOCUMENT NUMBER:     PubMed ID: 12465063
TITLE:               Diagnosing multiple system
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atrophy with greater accuracy: combined analysis of the clonidine-growth hormone test and external anal sphincter electromyography.

AUTHOR: Lee Eun Ah; Kim B Joon; Lee Won Yong

CORPORATE SOURCE: Department of Neurology, Samsung Medical Center, Sungkyunkwan University School of Medicine, Seoul, Korea.

SOURCE: Movement disorders : official journal of the Movement Disorder Society, (2002 Nov) Vol. 17, No. 6, pp. 1242-7.
Journal code: 8610688. ISSN: 0885-3185. L-ISSN: 0885-3185.

PUB. COUNTRY: United States

DOCUMENT TYPE: (COMPARATIVE STUDY)
(EVALUATION STUDIES)
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200303

ENTRY DATE: Entered STN: 17 Dec 2002
Last Updated on STN: 25 Mar 2003
Entered Medline: 24 Mar 2003

AB The clonidine-growth hormone test (CGHT) has been proposed as a means of differentiating multiple system atrophy (MSA) from idiopathic Parkinson's disease (PD). However, it is controversial whether the CGHT is valid. We sought to confirm the validity of the CGHT and to compare the diagnostic accuracy of the CGHT with that of external anal sphincter electromyelography (Sph-EMG) for MSA. We performed the CGHT and the Sph-EMG on 21 PD patients, 23 patients with probable MSA of parkinsonian type (MSA-p), and 22 patients with probable MSA of cerebellar type (MSA-c). We compared the sensitivity, specificity, and positive and negative predictive values (PPV and NPV) of CGHT, Sph-EMG, and a combination of the two tests. We also evaluated the correlations of Unified Parkinson's Disease Rating Scale (UPDRS) scores with the results of the two tests. There was no significant difference between the UPDRS scores for the PD and MSA-p groups. Serum growth hormone concentrations after clonidine significantly increased in PD (mean increase +/- SEM, 4.19 +/- 0.92 ng/ml; P < 0.0001), but remained unchanged in both MSA-p (0.83 +/- 0.61 ng/ml) and MSA-c (1.45 +/- 0.58 ng/ml). The growth hormone responses to clonidine in MSA-p were significantly different from those in PD (P < 0.05). Abnormal, denervated Sph-EMG was observed in 95.7% of MSA-p, 86.4% of MSA-c, and 33.3% of PD patients. Compared to Sph-EMG, the CGHT was less sensitive but more specific in both MSA-p and MSA-c. The result of neither test correlated with the severity of parkinsonism. Interestingly, combining the results of the CGHT and Sph-EMG markedly increased the specificity (85.7% in the CGHT and 66.7% in Sph-EMG vs. 95.2% in the combination study) and the PPV in both MSA-p (85.7% and 75.9% vs. 94.4%) and MSA-c (82.4% and 73.1% vs. 91.7%). We confirm that the CGHT can distinguish MSA-p from PD. Its sensitivity is lower and its specificity higher than Sph-EMG. Compared to either test alone, combined testing with the CGHT and Sph-EMG increased specificity and PPV, thereby enhancing accuracy in the diagnosis of MSA.

L6 ANSWER 2 OF 17 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 2002480153 MEDLINE

DOCUMENT NUMBER: PubMed ID: 12242540

TITLE: Stimulation of growth-hormone release with clonidine does not distinguish individual cases of idiopathic Parkinson's disease from those with striatonigral degeneration.

AUTHOR: Strijks E; van't Hof M; Sweep F; Lenders J W; Oyen W J; Horstink M W I M

CORPORATE SOURCE: Dept. of Neurology, University Medical Center, PO Box 9101, 6500 HB Nijmegen, The Netherlands.
SOURCE: Journal of neurology, (2002 Sep) Vol. 249, No. 9, pp. 1206-10.
Journal code: 0423161. ISSN: 0340-5354. L-ISSN: 0340-5354.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 21 Sep 2002
Last Updated on STN: 28 Dec 2002
Entered Medline: 27 Dec 2002

AB Multiple System Atrophy (MSA) and idiopathic Parkinson's disease (PD) can be difficult to distinguish. There is an ongoing debate about the diagnostic value of the growth-hormone response to clonidine (CGH-test) in PD and MSA. We investigated whether the CGH-test can identify individual patients in the early stages of PD (n = 21) and Striatonigral Degeneration (SND, n = 11), a particular variety of MSA. Patients were diagnosed on the basis of clinical criteria and IBZM-SPECT. Clonidine induced a greater total serum growth-hormone production in PD than in SND (p = 0.01). However, taking the difference in prevalence of PD and SND into account, and because of the low likelihood ratios of the test, an increase of GH after clonidine increases the pre-test probability for PD by about only 5 %, while an absent response of GH also increases the pre-test probability for SND by about 5 %. We conclude that the CGH-test discriminates between groups of patients with PD and SND, but has little practical diagnostic value for identifying individual patients.

L6 ANSWER 3 OF 17 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2002229887 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11967661
TITLE: Is clonidine-growth hormone stimulation a good test to differentiate multiple system atrophy from idiopathic Parkinson's disease?.
AUTHOR: Mathias C J; Kimber J; Watson L; Muthane U
SOURCE: Journal of neurology, (2002 Apr) Vol. 249, No. 4, pp. 488-9.
Journal code: 0423161. ISSN: 0340-5354. L-ISSN: 0340-5354.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Commentary
Letter
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200212
ENTRY DATE: Entered STN: 23 Apr 2002
Last Updated on STN: 27 Dec 2002
Entered Medline: 24 Dec 2002

L6 ANSWER 4 OF 17 MEDLINE on STN DUPLICATE 4
ACCESSION NUMBER: 2001171170 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11176962
TITLE: Increased growth hormone response to apomorphine in Parkinson disease compared with multiple system atrophy.
AUTHOR: Friess E; Kuempfel T; Winkelmann J; Schmid D; Uhr M; Rupprecht R; Holsboer F; Trenkwalder C
CORPORATE SOURCE: Max Planck Institute of Psychiatry, Kraepelinstr 10, D-80804 Munich, Germany.. friess@mpipsykl.mpg.de

SOURCE: Archives of neurology, (2001 Feb) Vol. 58, No. 2, pp. 241-6.
Journal code: 0372436. ISSN: 0003-9942. L-ISSN: 0003-9942.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200103
ENTRY DATE: Entered STN: 4 Apr 2001
Last Updated on STN: 4 Apr 2001
Entered Medline: 29 Mar 2001

AB BACKGROUND: Parkinson disease (PD) is often difficult to distinguish from parkinsonian syndromes of other causes in early stages of the disease. In search of a suitable endocrinologic challenge test, we investigated dopaminergic sensitivity in patients with de novo parkinsonian syndromes. OBJECTIVE: We measured the growth hormone (GH) response to a subthreshold dose of the dopamine 1-dopamine 2 receptor agonist apomorphine hydrochloride to differentiate parkinsonian syndromes from PD. PATIENTS AND METHODS: Seventeen patients with a clinical diagnosis of PD, 16 patients with a clinical diagnosis of multiple system atrophy, and 11 healthy controls. The GH response to a subthreshold dosage of apomorphine and to somatorelin (GH-releasing factor) was tested in a randomized order; on the third day the protocol was repeated with a clinically effective dose of apomorphine. RESULTS: The GH response to the low dose of apomorphine was significantly increased in patients with PD when compared with patients with multiple system atrophy or the control subjects (multivariate analyses of covariance; univariate F test, all P<.05). In contrast, there were no significant group differences with use of the higher dose of apomorphine or in the somatorelin-induced GH release. CONCLUSIONS: The GH response to a subthreshold dose of apomorphine appears to be a useful tool to identify patients with PD vs multiple system atrophy. The enhanced GH response to a subthreshold dopaminergic stimulus may reflect a hypersensitivity of the extrastriatal dopamine receptors in PD.

L6 ANSWER 5 OF 17 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2001440640 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11487211
TITLE: Stimulation of growth hormone release in multiple system atrophy, Parkinson's disease and idiopathic cerebellar ataxia.
AUTHOR: Pellecchia M T; Salvatore E; Pivonello R; Faggiano A; Barone P; De Michele G; Colao A M; Filla A
CORPORATE SOURCE: Department of Neurological Sciences, University Federico II, Naples, Italy.
SOURCE: Neurological sciences : official journal of the Italian Neurological Society and of the Italian Society of Clinical Neurophysiology, (2001 Feb) Vol. 22, No. 1, pp. 79-80.
Journal code: 100959175. ISSN: 1590-1874. L-ISSN: 1590-1874.
PUB. COUNTRY: Italy
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 13 Aug 2001
Last Updated on STN: 21 Jan 2002
Entered Medline: 13 Dec 2001

AB Clonidine has been proposed to differentiate multiple system atrophy (MSA) from idiopathic Parkinson's disease (IPD), as it does not increase growth hormone (GH) release in MSA. We studied GH release in response to clonidine in 7 IPD patients, 6 MSA patients, 4 patients affected by idiopathic late-onset cerebellar ataxia (ILOCA) and 8 healthy controls. In addition, we investigated the effects of GH releasing hormone plus arginine (GHRH-Arg) on GH release in the same patients. Both clonidine and GHRH-Arg raised serum GH levels in all groups examined. Clonidine failed to differentiate MSA from IPD and ILOCA. GHRH-Arg showed a lower increase of serum GH in MSA patients than in other groups, even if such difference was not statistically significant. We suggest that stimulation of GH release with GHRH-Arg rather than clonidine could differentiate MSA from IPD and ILOCA, but this hypothesis would need to be confirmed by further investigations.

L6 ANSWER 6 OF 17 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2000384832 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10869054
TITLE: Physiological, pharmacological and neurohormonal assessment of autonomic function in progressive supranuclear palsy.
AUTHOR: Kimber J; Mathias C J; Lees A J; Bleasdale-Barr K; Chang H S; Churchyard A; Watson L
CORPORATE SOURCE: Autonomic Unit, University Department of Clinical Neurology, Institute of Neurology, University College London, National Hospital for Neurology and Neurosurgery and Neurovascular Medicine Unit, London, UK.
SOURCE: Brain : a journal of neurology, (2000 Jul) Vol. 123 (Pt 7), pp. 1422-30.
Journal code: 0372537. ISSN: 0006-8950. L-ISSN: 0006-8950.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 18 Aug 2000
Last Updated on STN: 18 Aug 2000
Entered Medline: 7 Aug 2000

AB The clinical features of progressive supranuclear palsy (PSP) overlap with other parkinsonian syndromes, including multiple system atrophy (MSA). Autonomic dysfunction is a characteristic of MSA, but has also been described in PSP. We therefore report results from a series of physiological studies of cardiovascular autonomic function in 35 PSP and 20 MSA subjects, and 26 age-matched healthy control subjects. The response to growth hormone-clonidine testing, a neuropharmacological assessment of central adrenoceptor function, was also assessed in 14 PSP and 10 MSA subjects, and compared with 10 controls. None was on medication which may have affected the results. Orthostatic hypotension did not occur in PSP subjects or controls, unlike MSA subjects. Overall there was no evidence of sympathetic vasoconstrictor failure in PSP subjects, unlike MSA subjects, although the pressor response to mental arithmetic was reduced. Cardiac parasympathetic function was affected in only a minority (three of 35) of PSP subjects and was abnormal in MSA subjects. After clonidine administration, growth hormone rose in PSP subjects (median increase 4.3; interquartile range 1.8-7.8 mU/l) and controls, unlike MSA subjects (0.9; 0.3-2.4 mU/l; $P < 0.005$, Mann-Whitney U-test). In conclusion, in PSP subjects, responses to both physiological and pharmacological tests provided evidence against widespread autonomic dysfunction; this differed markedly from MSA subjects. Thus, cardiovascular autonomic dysfunction

should be an exclusionary feature in the diagnosis of PSP.

L6 ANSWER 7 OF 17 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2001190915 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11151417
TITLE: Is clonidine growth hormone stimulation a good test to differentiate multiple system atrophy from idiopathic Parkinson's disease?.
AUTHOR: Tranchant C; Guiraud-Chaumeil C; Echaniz-Laguna A; Warter J M
CORPORATE SOURCE: Service des Maladies du Systeme Nerveux et du Muscle, Hopitaux Universitaires, 1 Place de l'Hopital, 67091 Strasbourg, France. christine. tranchant@chru-strasbourg.fr
SOURCE: Journal of neurology, (2000 Nov) Vol. 247, No. 11, pp. 853-6.
Journal code: 0423161. ISSN: 0340-5354. L-ISSN: 0340-5354.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 10 Apr 2001
Last Updated on STN: 10 Apr 2001
Entered Medline: 5 Apr 2001
AB Clonidine, a centrally active alpha 2-adrenoreceptor agonist used to lower blood pressure, has been proposed to differentiate central from peripheral autonomic deficits and multiple system atrophy (MSA) from untreated idiopathic Parkinson's disease (IPD). A lack of growth hormone (GH) increase after clonidine infusion is found in patients with MSA, but not in those with IPD or with pure autonomic failure. We studied 19 IPD and 7 MSA patients to assess whether this test could be used in clinical practice to distinguish MSA from IPD, whatever the stage of the disease. Serum GH levels were measured 15, 30, 45 and 60 min after a 10-min infusion of 2 micrograms/kg clonidine. GH levels remained stable after clonidine infusion in all 7 MSA patients but increased in only 12 of the 19 IPD patients, while remaining stable in the other 7. No correlation was found with the presence of orthostatic hypotension. We conclude that the GH response to clonidine infusion has a very high sensitivity (100% in our series and in previous studies) for the diagnosis of MSA. However, this response cannot be used as a diagnostic test because of its poor specificity.

L6 ANSWER 8 OF 17 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 1999232905 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10218537
TITLE: Failure of the clonidine growth hormone stimulation test to differentiate multiple system atrophy from early or advanced idiopathic Parkinson's disease.
AUTHOR: Clarke C E; Ray P S; Speller J M
SOURCE: Lancet, (1999 Apr 17) Vol. 353, No. 9161, pp. 1329-30.
Journal code: 2985213R. ISSN: 0140-6736. L-ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Letter
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199905
ENTRY DATE: Entered STN: 1 Jun 1999
Last Updated on STN: 3 Mar 2000

Entered Medline: 20 May 1999

L6 ANSWER 9 OF 17 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 2000049368 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10584673
TITLE: Neuroendocrine responses to levodopa in multiple system atrophy (MSA).
AUTHOR: Kimber J; Watson L; Mathias C J
CORPORATE SOURCE: Division of Neuroscience and Psychological Medicine, Imperial College School of Medicine at St. Mary's Hospital, London, UK.
SOURCE: Movement disorders : official journal of the Movement Disorder Society, (1999 Nov) Vol. 14, No. 6, pp. 981-7.
Journal code: 8610688. ISSN: 0885-3185. L-ISSN: 0885-3185.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200001
ENTRY DATE: Entered STN: 31 Jan 2000
Last Updated on STN: 31 Jan 2000
Entered Medline: 20 Jan 2000

AB Hypothalamic dopaminergic pathways are involved in the regulation of growth hormone and prolactin release from the anterior pituitary. Neuroendocrine studies in patients with multiple system atrophy (MSA), in whom there is a reported loss of hypothalamic dopamine, are few and contradictory. We therefore studied the neuroendocrine responses to 250 mg levodopa (plus 25 mg carbidopa) in subjects with MSA (n = 15), and compared them with age- and sex-matched healthy control subjects (n = 8). There were no significant differences in basal or post-levodopa levels of growth hormone (GH), growth hormone-releasing hormone (GHRH), glucose, insulin-like growth factor (IGF-1), or thyroid-stimulating hormone (TSH) between the groups. In patients with MSA, basal levels of prolactin were elevated (21.1 +/- 5.2 ng/mL [mean +/-standard error]) compared with control subjects (12.1 +/- 1.7, p <0.05), and after L-dopa there was increased variability in prolactin response with less suppression compared with control subjects. In conclusion, in patients with MSA, the GHRH and GH responses to L-dopa were preserved and were similar to responses in age-matched control subjects. In contrast, there was impaired dopaminergic suppression of prolactin secretion. In patients with MSA this may represent a selective dysfunction, rather than generalized loss, of tubero-infundibular dopaminergic neurones.

L6 ANSWER 10 OF 17 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
ACCESSION NUMBER: 1998:290561 BIOSIS
DOCUMENT NUMBER: PREV199800290561
TITLE: Growth hormone (GH) secretion during sleep is similar in multiple system atrophy (MSA) and Parkinson's disease (PD).
AUTHOR(S): Pierangeli, Giulia; Barletta, Giorgio; Provini, Federica; Plazzi, Giuseppe; Maltoni, Paolo; Pavani, Anna; Bozza, Daniela; Lugaresi, Elio; Cortelli, Pietro
CORPORATE SOURCE: Bologna, Italy
SOURCE: Neurology, (April, 1998) Vol. 50, No. 4 SUPPL. 4, pp. A240-A241. print.
Meeting Info.: 50th Annual Meeting of the American Academy of Neurology. Minneapolis, Minnesota, USA. April 25-May 2, 1998. American Academy of Neurology.

CODEN: NEURAI. ISSN: 0028-3878.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 8 Jul 1998
Last Updated on STN: 8 Jul 1998

L6 ANSWER 11 OF 17 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 1998446275 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9773098
TITLE: [Pharmacologic approach to autonomic failure].
Approche pharmacologique des dysautonomies.
AUTHOR: Senard J M; Montastruc J L
CORPORATE SOURCE: Laboratoire de Pharmacologie Medicale et Clinique, INSERM U 317, Faculte de Medecine, Toulouse, France.
SOURCE: Therapie, (1998 Jan-Feb) Vol. 53, No. 1, pp. 35-41. Ref: 80
Journal code: 0420544. ISSN: 0040-5957. L-ISSN: 0040-5957.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (ENGLISH ABSTRACT)
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199811
ENTRY DATE: Entered STN: 6 Jan 1999
Last Updated on STN: 6 Jan 1999
Entered Medline: 3 Nov 1998
AB Four different forms of primary autonomic failure (multiple system atrophy, pure autonomic failure, Parkinson's disease and dopamine beta-hydroxylase deficiency) have been described. The first part of the article will focus on the interest to pharmacology of elucidating pathophysiological mechanisms underlying autonomic involvement at the central level (growth hormone response to clonidine acute challenge), presynaptic level (plasma catecholamine levels after yohimbine administration) and on post-synaptic receptors (binding studies, pressor responses to noradrenaline). The second part will discuss efficacy and side-effects of some of the many drugs which are currently proposed for the treatment of one of the most disabling symptoms related to autonomic failure, orthostatic hypotension. Special attention will be paid to drugs acting on blood composition (fludrocortisone, erythropoietin), on post-synaptic alpha-adrenoceptors (midodrine and clonidine) and on noradrenaline spill-over (yohimbine and L-Threo-DOPS).

L6 ANSWER 12 OF 17 MEDLINE on STN DUPLICATE 11
ACCESSION NUMBER: 1997360797 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9217760
TITLE: Distinction of idiopathic Parkinson's disease from multiple-system atrophy by stimulation of growth-hormone release with clonidine.
AUTHOR: Kimber J R; Watson L; Mathias C J
CORPORATE SOURCE: University Department of Clinical Neurology, National Hospital for Neurology and Neurosurgery/Institute of Neurology, London, UK.
SOURCE: Lancet, (1997 Jun 28) Vol. 349, No. 9069, pp. 1877-81.
Journal code: 2985213R. ISSN: 0140-6736. L-ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (COMPARATIVE STUDY)

Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199707
ENTRY DATE: Entered STN: 12 Aug 1997
Last Updated on STN: 12 Aug 1997
Entered Medline: 28 Jul 1997

AB BACKGROUND: Idiopathic Parkinson's disease is a common neurodegenerative disease that is difficult to distinguish from other parkinsonian syndromes such as multiple-system atrophy (MSA). In MSA, autonomic dysfunction is common and is associated with either parkinsonian or cerebellar features, or both. Differentiation of idiopathic Parkinson's disease from MSA is important because prognosis, complications, and response to therapy vary according to disorder. Our aim was to find out whether clonidine/growth hormone (GH) testing distinguishes idiopathic Parkinson's disease from MSA. METHODS: Clonidine is a centrally active alpha 2-adrenoceptor agonist that raises concentrations of GH in serum in healthy people and those with pure autonomic failure (with peripheral lesions), but not in those with MSA (with a central autonomic deficit). We investigated the effects of clonidine on 14 people with idiopathic Parkinson's disease (without autonomic deficits). 31 people with MSA of the three different clinical forms (parkinsonian, cerebellar, and mixed), 19 people with pure autonomic failure, and 27 healthy participants. In nine people with parkinsonian MSA (MSA-P), the GH response to levodopa was also assessed. FINDINGS: Clonidine raised serum GH concentrations in patients with idiopathic Parkinson's disease (median increase 8.98 [IQR 6.6-16.6] mU/L), normal participants (13.2 [7.0-18.6] mU/L), and patients with pure autonomic failure (12.5 [5.6-18.2] mU/L). In those with MSA who had central autonomic failure, GH concentrations were unchanged (MSA-P; 0.41 [-0.30 to 2.09] mU/L and cerebellar MSA [MSA-C] 1.67 [0-4.49] mU/L). The GH response to clonidine in idiopathic Parkinson's disease was significantly different from that in MSA-P ($p < 0.0002$). In MSA-P, the dopamine precursor levodopa raised GH concentrations (from mean 2.7 [SE 1.0] mU/L to mean 18.2 [6.0] mU/L, $p < 0.05$) and GH-releasing hormone (GHRH) concentrations (from mean 20.6 [3.25] ng/L to mean 68.0 [10.6] ng/L, $p < 0.05$), excluding dysfunction of pituitary somatotrophs or GHRH neurons as a cause for the absent GH response to clonidine in MSA. INTERPRETATION: The GH responses to clonidine clearly differentiated idiopathic Parkinson's disease from MSA-C and MSA-P. Together with the levodopa studies they indicated a specific alpha 2-adrenoceptor-hypothalamic deficit in MSA. The clonidine-GH test may provide further insight into central neurotransmitter and alpha 2-adrenoceptor-hypothalamic abnormalities in MSA.

L6 ANSWER 13 OF 17 MEDLINE on STN DUPLICATE 12
ACCESSION NUMBER: 1997054962 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8899251
TITLE: Neurohumoral, peptidergic and biochemical responses to supine exercise in two groups with primary autonomic failure: Shy-Drager syndrome/multiple system atrophy and pure autonomic failure.
AUTHOR: Smith G D; Watson L P; Mathias C J
CORPORATE SOURCE: Department of Medicine, St Mary's Hospital medical School/Imperial College of Science, London, UK.
SOURCE: Clinical autonomic research : official journal of the Clinical Autonomic Research Society, (1996 Oct) Vol. 6, No. 5, pp. 255-62.
Journal code: 9106549. ISSN: 0959-9851. L-ISSN: 0959-9851.

PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 5 Mar 1997
Last Updated on STN: 5 Mar 1997
Entered Medline: 19 Feb 1997

AB The neurohumoral, peptidergic and biochemical responses to supine leg exercise were studied in two groups with primary autonomic failure: Shy-Drager syndrome (SDS, n = 15) and pure autonomic failure (PAF, n = 15), to determine if these accounted for exercise-induced hypotension and the greater blood pressure (BP) fall in PAF. Responses were compared to normal subjects (controls, n = 15), in whom BP rose with exercise. Resting plasma noradrenaline (NA) was higher in controls than SDS, and was lowest in PAF. With exercise, NA increased in controls, with a small rise in SDS, but no change in PAF. Resting plasma adrenaline (A) was higher in controls and SDS than PAF, with no change during exercise. Plasma dopamine was unrecordable at all stages in all groups. Resting plasma renin activity (PRA) was higher in controls than SDS and PAF, and was unchanged with exercise in all groups. Plasma insulin, C-peptide and serum growth hormone (GH) were similar at
rest and with exercise in the three groups. Plasma glucose was higher at rest in SDS and PAF, and increased with exercise in all three groups. In conclusion, neither exercise-induced hypotension, nor the differences between SDS and PAF could be related to abnormalities in the release of A, PRA, insulin, glucose or GH. The abnormal NA response to exercise was consistent with the BP fall being due to inadequate compensatory sympathetic activity. In SDS, the small NA increase, in the presence of supersensitivity, may have reduced their BP fall as compared to PAF. These results suggest that impaired sympathetic neural activity is a key factor in exercise-induced hypotension.

L6 ANSWER 14 OF 17 BIOSIS COPYRIGHT (c) 2010 The Thomson Corporation on STN
ACCESSION NUMBER: 1996:446224 BIOSIS
DOCUMENT NUMBER: PREV199699168580
TITLE: Neuropharmacological evaluation of hypothalamic alpha-adrenoceptor deficit in human central sympathetic degeneration.
AUTHOR(S): Kimber, J. [Reprint author]; Watson, L.; Mathias, C. J.
CORPORATE SOURCE: Autonomic Unit, Univ. Dep. Clinical Neurol., Inst. Neurol., Queen Square, UK
SOURCE: Journal of Physiology (Cambridge), (1996) Vol. 494P, No. 0, pp. 138P-139P.
Meeting Info.: Scientific Meeting of the Physiological Society. London, England, UK. April 16-18, 1996.
CODEN: JPHYA7. ISSN: 0022-3751.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 7 Oct 1996
Last Updated on STN: 7 Oct 1996

L6 ANSWER 15 OF 17 MEDLINE on STN DUPLICATE 13
ACCESSION NUMBER: 1995199869 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7892755
TITLE: High beta-adrenoceptor density on peripheral blood mononuclear cells in progressive multiple sclerosis: a manifestation of autonomic dysfunction?.

AUTHOR: Zoukos Y; Thomaides T; Mathias C J; Cuzner M L
CORPORATE SOURCE: Multiple Sclerosis Laboratory, National Hospital for Neurology and Neurosurgery, London, England.
CONTRACT NUMBER: (United Kingdom Wellcome Trust)
SOURCE: Acta neurologica Scandinavica, (1994 Dec) Vol. 90, No. 6, pp. 382-7.
Journal code: 0370336. ISSN: 0001-6314. L-ISSN: 0001-6314.
PUB. COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199504
ENTRY DATE: Entered STN: 27 Apr 1995
Last Updated on STN: 29 Jan 1999
Entered Medline: 20 Apr 1995

AB In multiple sclerosis (MS) up-regulation of beta-adrenoceptors on peripheral blood mononuclear cells (PBMCs) has been attributed to either autonomic dysfunction, inflammation or a combination of the two. We have compared secondary progressive MS patients with normal subjects (NS) and two models of autonomic dysfunction; pure autonomic failure (PAF) and multiple system atrophy (MSA, Shy-Drager syndrome). There was up-regulation of beta-adrenoceptors on PBMCs in MS and PAF patients but not in MSA patients. Only in PAF patients beta-adrenoceptor up-regulation was correlated with low plasma levels of noradrenaline (NA) and adrenaline (Ad). In addition to studies in the basal state, measurements also were made after the centrally acting sympatholytic agent clonidine. These were combined with haemodynamic and neurohormonal measurements. After clonidine, there was a fall in blood pressure in NS and MSA patients but not in MS and PAF patients; a rise in growth hormone (GH) in NS and PAF patients but not in MS and MSA patients; and an up-regulation in PBMCs beta-adrenoceptors in NS but not in MS, MSA and PAF patients. Up-regulation of beta-adrenoceptors on PBMCs in MS could be attributed to autonomic dysfunction but the disparity between MS and PAF patients when considering their plasma levels of NA and Ad argue against. Although the neurohormonal responses to clonidine and the physiological assessment of autonomic function in progressive MS patients, demonstrate central autonomic dysfunction resembling that of the MSA patients, the normal basal beta-adrenoceptor densities in the latter, suggests that the up-regulation of these receptors is independent of the central autonomic dysfunction in MS.

L6 ANSWER 16 OF 17 MEDLINE on STN DUPLICATE 14
ACCESSION NUMBER: 1994224346 MEDLINE
DOCUMENT NUMBER: PubMed ID: 8170565
TITLE: Beta-adrenoceptor expression on circulating mononuclear cells of idiopathic Parkinson's disease and autonomic failure patients before and after reduction of central sympathetic outflow by clonidine.
AUTHOR: Zoukos Y; Thomaides T; Pavitt D V; Cuzner M L; Mathias C J
CORPORATE SOURCE: Department of Neurochemistry, National Hospital for Neurology and Neurosurgery, London, UK.
CONTRACT NUMBER: (United Kingdom Wellcome Trust)
SOURCE: Neurology, (1993 Jun) Vol. 43, No. 6, pp. 1181-7.
Journal code: 0401060. ISSN: 0028-3878. L-ISSN: 0028-3878.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199406

ENTRY DATE: Entered STN: 13 Jun 1994
Last Updated on STN: 13 Jun 1994
Entered Medline: 2 Jun 1994

AB There is a short-term up-regulation of beta-adrenoceptors on peripheral blood mononuclear cells (PBMC) after reduction of central sympathetic outflow by clonidine in normal individuals. We have studied beta-adrenoceptor number and affinity on PBMC in idiopathic Parkinson's disease (PD), pure autonomic failure (PAF), and multiple system atrophy (MSA; Shy-Drager syndrome) patients and age- and sex-matched normal controls (NC) before and after intravenous administration of clonidine, an alpha 2-adrenoceptor agonist which lowers blood pressure predominantly by reducing CNS sympathetic outflow. Basal beta-adrenoceptor density was high in PAF but within the normal range in PD and MSA patients. After clonidine there was a decrease in plasma levels of noradrenaline (NA) and adrenaline (Ad) in PD, MSA, and NC, and an increase in growth hormone (GH) in PD, PAF, and NC. NC. In PAF, NA and Ad remained unchanged. In MSA, there was no increase in GH levels. There was an up-regulation of beta-adrenoceptors on PBMC at 30 and 60 minutes after clonidine administration, which returned to baseline values after 2 hours, and the affinity of the receptors was decreased in NC and PD patients. Intracellular production of cAMP after isoproterenol stimulation demonstrated that the up-regulation was not functional. Up-regulation after clonidine did not occur in PAF and MSA patients. The observed correlation of plasma NA and sympathetic defect with basal and clonidine-induced up-regulation of beta-adrenoceptors on PBMC may provide insight into beta-adrenoceptor changes in other tissues and also help in differentiating subgroups of autonomic failure patients.

L6 ANSWER 17 OF 17 MEDLINE on STN DUPLICATE 15
ACCESSION NUMBER: 1992341842 MEDLINE
DOCUMENT NUMBER: PubMed ID: 1353191
TITLE: Growth hormone response to clonidine in central and peripheral primary autonomic failure.
AUTHOR: Thomaides T N; Chaudhuri K R; Maule S; Watson L; Marsden C D; Mathias C J
CORPORATE SOURCE: Department of Medicine, St Mary's Hospital Medical School, Imperial College of Science, Technology and Medicine, London, UK.
CONTRACT NUMBER: (United Kingdom Wellcome Trust)
SOURCE: Lancet, (1992 Aug 1) Vol. 340, No. 8814, pp. 263-6.
Journal code: 2985213R. ISSN: 0140-6736. L-ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199208
ENTRY DATE: Entered STN: 11 Sep 1992
Last Updated on STN: 6 Feb 1995
Entered Medline: 25 Aug 1992

AB Patients with primary autonomic failure may have either pure autonomic failure (PAF) or multiple system atrophy (MSA) in which there is additional neurological involvement. Distinction between PAF and MSA at an early stage is important because a wide range of complications is associated with MSA, which has a poor response to drug therapy and a less favourable prognosis. We have investigated the growth hormone (GH) releasing effects of clonidine in patients with PAF and MSA to see whether this hormonal response could serve as a neuroendocrine marker to distinguish between the groups. Age-matched normal subjects were studied as controls. Both

groups of patients had severe postural hypotension due to primary sympathetic failure of presumed central origin in MSA and peripheral origin in PAF. After clonidine, plasma GH concentrations increased in controls and PAF, with no change in MSA. Changes in plasma glucose and insulin concentrations were similar in all groups. Clonidine, therefore, stimulates growth hormone release in PAF but not MSA and may serve as a neuroendocrine marker in differentiating patients with MSA and a central autonomic defect from those with PAF with a peripheral defect.

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ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

     (FILE 'HOME' ENTERED AT 12:13:35 ON 11 MAY 2010)

     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:13:43 ON 11 MAY 2010
L1          262 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  (GROWTH(W) HORMONE OR GH
                OR HGH) AND (MULTIPLE(W) SYSTEM(W) ATROPHY OR MSA)
L2           96 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  (GROWTH(W) HORMONE OR GH
                OR HGH) AND MULTIPLE(W) SYSTEM(W) ATROPHY
L3            4 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  L2 AND (SUBCUTANEOUS OR
                INTRAMUSC?)
L4            3 DUP REM L3 (1 DUPLICATE REMOVED)
L5           46 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  L2 AND PY<2003
L6           17 DUP REM L5 (29 DUPLICATES REMOVED)
                DIS IBIB ABS L4 1-3

     FILE 'STNGUIDE' ENTERED AT 12:17:30 ON 11 MAY 2010

     FILE 'MEDLINE, BIOSIS' ENTERED AT 12:19:58 ON 11 MAY 2010
                DIS IBIB ABS L6 1-17

     FILE 'STNGUIDE' ENTERED AT 12:19:59 ON 11 MAY 2010
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.56      67.66

STN INTERNATIONAL LOGOFF AT 12:24:59 ON 11 MAY 2010
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